Exhibit 5

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Date: November 3, 2025

Jerad J. Miller
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Re: Expert Rebuttal Opinion of Dr. Marehbian to the report of Dr. Omalu in the matter of *Jennie Quan, individually and as successor in interest to Benjamin Chin, deceased, v. County of Los Angeles, Marisol Barajas, et al;* Case No.: 2:24-cv-04805-MCS-KS

Date of Birth: January 6, 1993
Date of Incident: June 19, 2023

Dear Mr. Miller,

Please find attached my expert rebuttal regarding the opinions expressed by Dr. Bennet Omalu, in his report dated October 18, 2025, in the above-referenced matter. This report specifically addresses Dr. Omalu's conclusions concerning the presence and duration of conscious pain and suffering experienced by Mr. Benjamin Chin. My analysis is limited strictly to the scope of rebutting Dr. Omalu's report and does not introduce any new independent opinions or alternative causation theories.

All conclusions expressed in the attached rebuttal are stated to a reasonable degree of medical certainty and are based on my detailed review of the materials listed below. I have not conducted any independent medical examinations. My role in this matter is that of an expert medical reviewer, evaluating the scientific validity and internal consistency of Dr. Omalu's findings within the framework of established neurological and critical care principles.

If additional records, data, or materials become available, I reserve the right to amend, supplement, or modify my opinions accordingly to ensure that they remain accurate and complete.

Please do not hesitate to contact me should you require clarification or additional information.

Material Reviewed for This Report

- **Plaintiff's First Amended Complaint**
- **Plaintiff's Expert Disclosures and Reports:** Including Report of Dr. Bennet Omalu.
- **Decedent's Medical Records:** Pomona Valley Hospital Medical Center records.
- **Pathology/Forensic Documents:** Autopsy Report of Benjamin Chin.
- **Investigative Materials:**
    - Quan Homicide File (including supplemental reports of responding deputies).
    - Supplemental reports of responding deputies
    - Body-worn camera footage of Deputy Barajas and Detective Vazquez sync'd and of a Deputy and EMS providing medical care to Decedent Benjamin Chin.
- **Prior Expert Opinions (For Context):**
    - Defense expert Ed Flosi's report re: police practices.
    - Defense expert Joel Suss' report re: human factors.

Jonathan Marehbian, MD.

Neurology and Emergency Neurology

NPI:1972875730

SUMMARY OF REVIEW OF MEDICAL RECORDS

On June 19, 2023, at approximately 11:36 a.m., law enforcement encountered the patient on Diamond Bar Boulevard following reports of an armed individual. A deputy-involved shooting occurred, after which Los Angeles County Fire Department personnel treated the patient at the scene and transported him to Pomona Valley Hospital Medical Center. He arrived as a Level 1 trauma activation with multiple gunshot wounds to the chest and abdomen, unresponsive and with only a weak carotid pulse.

Upon assessment in the trauma bay, the patient was documented with a profound neurological deficit. The physical examination noted the Glasgow Coma Scale (GCS) as 1/1/1, resulting in a total GCS score of 3/15. The General assessment stated the patient was "unresponsive". The extremity examination noted bilateral upper and lower extremities with "no deformities," "no pain on palpation/range of motion," and "cool with no distal pulses noted".

During attempted endotracheal intubation in the trauma bay, he lost pulses and went into cardiac arrest. A resuscitative clamshell thoracotomy was performed emergently with aortic cross-clamping, resulting in return of pulses. Massive transfusion protocol was initiated and tranexamic acid was administered. A right internal jugular cordis catheter was placed, he was mechanically ventilated, and vasopressor support with norepinephrine and vasopressin was started.

Initial laboratory studies showed profound shock physiology with severe metabolic derangements, including severe metabolic acidosis, anemia, thrombocytopenia, hypofibrinogenemia, markedly prolonged partial thromboplastin time, and hypocalcemia. The body temperature minimum was documented as 36.5 degrees C (Axillary). During the resuscitation and hospital course, the patient received continuous infusion of sedative and analgesic agents, including Propofol and Fentanyl. He was transported emergently to the operating room.

In the first operation that day, exploratory laparotomy revealed immediate hemoperitoneum, a Grade V laceration of the right lobe of the liver with devitalized parenchyma, and an actively bleeding laceration of the right kidney. Definitive hemorrhage control and damage-control procedures were undertaken, including right nephrectomy and hepatic repair with partial right hepatectomy. He was transferred to the Trauma Intensive Care Unit in critical condition, receiving ongoing blood product resuscitation and maximal vasopressor support.
Despite these measures, he remained profoundly unstable with persistent diffuse oozing suggestive of disseminated intravascular coagulation. He was returned to the operating room later the same day for re-exploration. Intraoperative findings included ongoing thin hemoperitoneum, continued oozing from hepatic edges with the prior repair intact, a diffusely bleeding retroperitoneum, and ischemic changes of the right colon and distal small bowel. A subtotal colectomy and an extensive small bowel resection were performed. He was returned to the intensive care unit in critical condition.

Following re-exploration, he received continued massive transfusion and maximal vasoactive support but developed refractory shock. He suffered a cardiac arrest in the ICU, and after multiple rounds of Advanced Cardiovascular Life Support, there was no return of spontaneous circulation. Death was pronounced at 11:09 p.m. on June 19, 2023.

Pathology of operative specimens confirmed traumatic disruption of benign renal and hepatic parenchyma, with no intrinsic lesions. Resections of colon and small bowel demonstrated marked ischemic injury with submucosal hemorrhage and perforation, consistent with intraoperative findings of mesenteric vascular thrombosis and shock-related mucosal compromise.

A forensic autopsy on June 28, 2023, documented two through-and-through gunshot wounds. The second wound, which produced a 20 x 9 x 8 cm laceration of the right hepatic lobe, was deemed rapidly fatal due to massive hemorrhage. Toxicology was consistent with therapeutic Fentanyl administration during resuscitation. The medical examiner concluded the cause of death was multiple gunshot wounds.

DISCUSSION

Dr. Bennet Omalu asserts that Benjamin Chin experienced conscious pain and suffering beginning at 11:36 a.m., when he first encountered law enforcement, continuing through the shooting at approximately 11:45 a.m., and lasting for a "composite mean, mode, and median period of less than 14 minutes," ending when "global hypoxic-ischemic brain injury began." This defined period forms the temporal backbone of his opinion and allows his reasoning to be tested against established neurological and physiological principles.

Video evidence confirms that Mr. Chin exhibited minimal responsiveness immediately following the shooting, consistent with a fleeting, pre-syncope state of reactive awareness. This brief window may have allowed transient nociceptive input, but cerebral perfusion and oxygenation were rapidly failing due to catastrophic hemorrhage. Within seconds, perfusion pressure would have fallen below the level required to sustain cortical activity. This phase represents a short-lived and physiologically collapsing period of potential awareness, not sustained or organized conscious suffering.

Dr. Omalu's assertion that compensable "mental, somatic, and biochemical pain and suffering" began at 11:36 a.m. is medically unsupportable. This portion of his timeline predates the physical injury and relies on presumed fear or apprehension. Fear is a psychological and emotional state, not an objectively measurable medical condition. By attempting to include pre-injury emotional distress within "suffering," Dr. Omalu moves outside the bounds of medical science and into speculative psychology. The compensable experience of pain can only begin at the moment of traumatic injury, approximately 11:45 a.m.

At 11:59 a.m., upon arrival at Pomona Valley Hospital Medical Center, Mr. Chin was documented as unresponsive with a Glasgow Coma Scale of 3/15. This finding represents the absolute clinical endpoint of consciousness. A GCS 3 reflects total absence of eye opening, verbal response, and motor command, signifying the loss of integrated cortical and thalamic function necessary for awareness. From this point forward, no meaningful sensory perception, cognition, or subjective experience could occur.

Dr. Omalu himself defines the boundary of consciousness when he writes that suffering ends when "global hypoxic-ischemic brain injury begins." That statement is accurate. However, his

report simultaneously suggests that Mr. Chin's conscious suffering may have continued for "less than 14 minutes" following the shooting and that "pain and suffering persisted until the complete cessation of all bodily functions, including cardiac and respiratory arrest." These positions are mutually incompatible.

Mr. Chin's autopsy and operative findings document a Grade V liver laceration, a wound explicitly described as rapidly fatal, producing immediate exsanguination and loss of cerebral perfusion. The biological window for any residual consciousness in such an injury is measured in minutes, not hours. Once cerebral oxygenation drops below the metabolic threshold for synaptic transmission, integrated cortical function ceases, marking the end of awareness. This conclusion is consistent with established medical understanding of brain physiology under traumatic shock.

The subsequent GCS 3 coma reflected complete central nervous system collapse due to hypovolemic shock, traumatic cardiac arrest, and anoxic encephalopathy. Laboratory data confirmed profound metabolic acidosis and coagulopathy, accompanied by lower body temperatures, consistent with ongoing circulatory collapse and conditions incompatible with sustained cerebral perfusion or awareness. These converging mechanisms abolished all neural network connectivity, including the frontoparietal and cingulo-insular systems required for conscious pain perception.

From that point forward, Mr. Chin's medical course was sustained exclusively by mechanical ventilation, transfusion, and pharmacologic support. He remained under continuous infusion of propofol and fentanyl, which ensured total suppression of cortical activity and nociceptive awareness. Propofol produces generalized cortical inhibition, while fentanyl blocks both sensory-discriminative and emotional-affective components of pain. Together they constitute a chemical and ethical guarantee against any form of consciousness, pain, or suffering. Thus, while circulation was mechanically supported for approximately 11 hours, this time represented biological maintenance of an unconscious body, not prolonged conscious suffering.

Dr. Omalu's report contains fundamental internal contradictions regarding the neuroanatomy of pain and suffering. He oscillates between two incompatible models, one acknowledging the necessity of cortical activity for conscious pain, and another asserting that reflexive or subcortical processes alone suffice.

Early in his report, Dr. Omalu correctly writes that:

> "The sensation of pain induces conscious suffering since pain is a noxious sensation which stimulates the neocortex, limbic cortex, and forebrain to cause mental pain and suffering."

This statement aligns with basic neurophysiology: conscious suffering requires intact cortical and limbic participation. Yet elsewhere, he abandons this definition to preserve his extended timeline of suffering. When faced with the fact that Mr. Chin was comatose, Dr. Omalu redefines pain as a primitive process:

> "Pain perception is principally a function of the lower centers of the brain such as the brainstem, hypothalamus, and limbic system, not necessarily of the cerebral cortex."

These two claims cannot coexist. If pain "induces conscious suffering" by engaging the neocortex and forebrain, then pain cannot persist once those structures are nonfunctional. By his own words, the requirement of cortical participation precludes the possibility of suffering in a comatose, anoxic brain. The GCS 3 finding and documented anoxic injury demonstrate that these structures were physiologically silent.

To justify his prolonged claim of suffering during coma, Dr. Omalu writes that "the spinal reflex is the foundational basis for pain and suffering." This statement reflects a categorical error. A reflex is a non-conscious motor or autonomic response originating in the spinal cord or brainstem. It indicates the presence of neural circuitry, not awareness. Movements such as grimacing, limb withdrawal, or hemodynamic fluctuations represent nociception, not conscious pain.

By redefining reflexive physiological responses as "suffering," Dr. Omalu collapses the distinction between unconscious neural activity and conscious experience. This maneuver allows the stretch of the duration of supposed suffering well beyond the limits of consciousness he himself defines. In effect, he substitutes reflex biology for conscious perception, thereby constructing an internally self-contradictory and scientifically incoherent model.

Dr. Omalu concludes that pain and suffering persist "until the complete cessation of all bodily functions, including cardiac and respiratory arrest." This is a metaphysical proposition, not a medical one. Pain and suffering are dependent on active neural processing. Once circulation and oxygenation to the brain cease, neurons depolarize and synaptic transmission halts within seconds. Without brain activity, there can be no perception, emotion, or awareness.

The objective record demonstrates that Mr. Chin experienced, at most, a short-lived period of conscious pain in the immediate aftermath of injury, lasting no more than a few minutes. Thereafter, he was in a state of irreversible coma, compounded by pharmacologic suppression and loss of cortical activity. The remaining 11 hours were characterized by mechanical and pharmacologic support of an unconscious patient who underwent repeated life-saving surgical interventions.

Dr. Omalu's opinion attributes nearly all of this survival time to "conscious suffering" based on a self-refuting redefinition of pain. His report simultaneously requires and denies cortical participation, treating reflexive activity as subjective experience. The result is a theory that contradicts itself at every critical juncture.

In summary, conscious pain was brief and biologically limited to the immediate post-injury period. Loss of awareness likely occurred earlier, during pre-hospital care, as suggested by the rapid physiological collapse observed at the scene, though confirmation awaits review of the EMS records. By 11:59 a.m., and almost certainly before that time, there was total loss of consciousness, confirmed by objective neurological findings and sustained pharmacologic coma. The claim of suffering persisting for 11 hours is medically indefensible, internally inconsistent, and unsupported by any known principles of neuroscience.

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